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WHAT'S NEW IN INTENSIVE CARE

Heart–lungs interactions in mechanically ventilated patients: physiology and clinical implications

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Hemodynamic alterations induced by mechanical ventilation result from complex interactions between cardiovascular and respiratory systems within the confined thoracic space. Unlike spontaneous breathing, mechanical insufflation generates positive alveolar and intrathoracic pressures throughout the respiratory cycle, significantly altering right-ventricular (RV) and left-ventricular (LV) loading conditions. Compared to physiological conditions, heart–lungs interactions are more marked during mechanical ventilation and in certain pathological situations, where they may have clinical implications [1].

Main cardiac effects of mechanical insufflation

Decrease in RV preload

Mechanical insufflation increases intrathoracic pressure (P_{IT}), and thus the right atrial pressure, reducing the venous return pressure gradient [2]. On the other hand, both increased intra-abdominal pressure (P_{IA}) due to diaphragmatic descent and passive transmission of P_{IT}, and baroreflex sympathetic activation, increase the upstream pressure of venous return. These compensatory mechanisms may be limited in cases of impaired baroreflex or hypovolemia, where increased P_{IA} may increase the resistance to venous return.

Increase in RV afterload

There is a U-shaped relationship between pulmonary vascular resistance (PVR) and lung volume, with a nadir at the functional residual capacity (FRC), which is the end-expiratory lung volume in normal conditions [3]. During mechanical insufflation, the transpulmonary pressure (P_{TP} = alveolar pressure—P_{IT}) increases, compressing the intra-alveolar vessels and, thus, increasing PVR and RV afterload. This increased P_{TP} during insufflation is amplified by higher tidal volume (V_T) ventilation, reduced lung compliance, and increased chest wall compliance [4]. Hypovolemia—which extends West's zone 2 at the expense of zone 3 through reduced pulmonary venous pressure—can also increase PVR and impair RV afterload [5].

Increase in LV preload

LV preload is increased during mechanical insufflation through two mechanisms. First, the increased P_{TP} induces blood transfer from pulmonary capillaries toward the left atrium. Second, the decrease in LV filling secondary to the inspiratory decrease in RV stroke volume (SV) (serial biventricular interdependence) occurs only during the following expiration due to the pulmonary transit time.

Heart-lungs interactions in mechanically ventilated patients are summarized in Fig. 1.

Decrease in LV afterload

LV afterload decreases during mechanical insufflation due to the transmission of the increased P_{IT} both to the left ventricle and the intrathoracic aorta, reducing the aortic transmural pressure [6].

Clinical implications

Prediction of fluid responsiveness

When both ventricles are preload-dependent, insufflation results in a decreased RV SV due to the decreased

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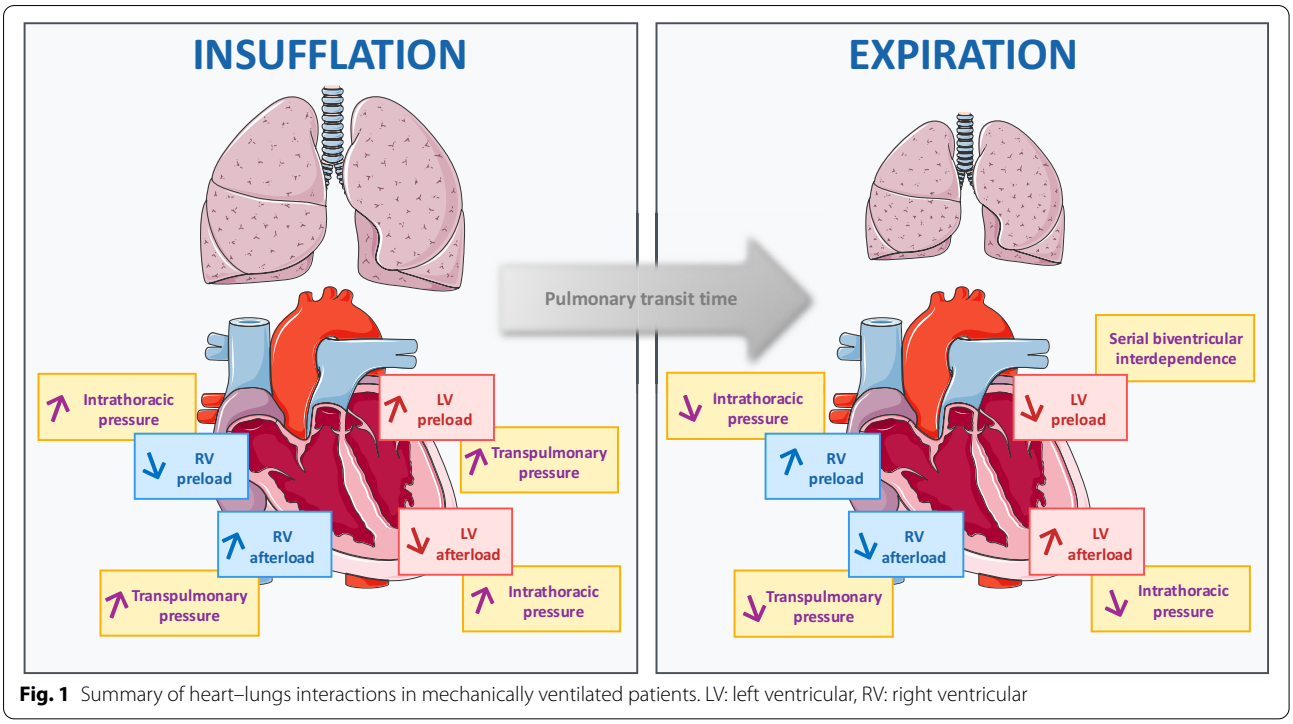

Fig. 1 Summary of heart–lungs interactions in mechanically ventilated patients. LV: left ventricular, RV: right ventricular

venous return, and a delayed decrease in LV SV during the following expiration owing to the pulmonary transit time. This results in marked respiratory changes in SV and, thus, in pulse pressure (PP). Since fluid responsiveness also occurs when both ventricles are preload-dependent, the magnitude of respiratory changes in PP (PPV) is assumed to reflect the degree of fluid responsiveness [7]. PPV has been widely validated as a predictor of fluid responsiveness [7] and is strongly recommended in recent guidelines when it is applicable [8]. Conditions of its inapplicability include spontaneous breathing activity, arrhythmias and low V_T (< 8 mL/kg) [7]. In the latter case, fluid responsiveness can be reliably assessed using PPV changes in response to transient V_T increase [9] or to passive leg raising [10]. Other heart–lung interactions tests such as end-expiratory occlusion can also be used to predict fluid responsiveness [8].

Weaning-induced pulmonary edema

The risk factors of weaning-induced pulmonary edema (WIPO) include chronic obstructive pulmonary disease and pre-existing cardiopathy [11]. During weaning from mechanical ventilation, the abrupt shift from a positive to a negative inspiratory P_{IT}, potentially amplified by endotracheal tube resistance, simultaneously increases RV and LV afterload and potentially results in WIPO [12] that may prevent successful weaning in predisposed patients. Understanding the underlying mechanism(s) is important to prevent or treat WIPO properly.

Cardiogenic pulmonary edema

Heart–lungs interactions explain the beneficial cardiovascular effects of positive pressure ventilation in patients with acute cardiogenic pulmonary edema [13]. Positive pressure ventilation reduces both the RV preload and the LV afterload due to the suppression of negative inspiratory P_{IT}, facilitating the LV ejection in case of failing left ventricle [14]. This is particularly relevant in spontaneously breathing patients, in whom positive pressure ventilation attenuates the negative inspiratory P_{IT} and its adverse effects by reducing the LV transmural pressure. Moreover, positive pressure ventilation reduces respiratory muscle oxygen consumption and improves coronary perfusion by decreasing LV end-diastolic pressure.

Acute respiratory distress syndrome

In patients with acute respiratory distress syndrome (ARDS), the cyclic changes in P_{IT} are small and expected to exert minor consequences on RV and LV afterload due to the reduced lung compliance and use of low V_T ventilation. Heart–lungs interactions mainly involve the effects of P_{TP} on RV afterload, although low V_T ventilation reduces the occurrence of RV dysfunction. Positive end-expiratory pressure (PEEP) induces variable effects on RV afterload, depending on its effects on the

lung recruitment/overdistension ratio [15] and according to the U-shaped relationship between PVR and lung volume [3]. If PEEP markedly recruits previously closed lung units, it will increase the end-expiratory lung volume toward FRC, so that PVR decreases a little and thus will not increase during low V_T insufflation. Conversely, if PEEP predominantly distends normal lung alveoli, it compresses intra-alveolar microvessels and thereby increases PVR before and even more during V_T inflation. This can induce RV dysfunction with uncoupling between the right ventricle and the pulmonary circulation [15]. A decreased central blood volume may reinforce the PEEP-induced increase in RV afterload, due to a larger proportion of lungs in West's zone 2 conditions [5]. Therefore, to prevent adverse effects of PEEP on RV function, it is important to ensure that (1) PEEP is predominantly lung recruitive rather than lung distending and (2) hypovolemia induced by a deliberate "dry strategy" is not marked. Preload responsiveness tests help identify such a phenomenon.

Heart–lung interactions during mechanical ventilation result from complex P_{IT} and P_{TP} changes, inducing a decrease in RV preload and in LV afterload, an increase in RV afterload, and in LV preload [1]. Heart–lung interactions mainly account for the hemodynamic effects of PEEP in ARDS, the occurrence of WIPO, and the beneficial effects of positive pressure ventilation in cardiogenic pulmonary edema, and can be used to predict fluid responsiveness.

Abbreviations
ARDS: Acute respiratory distress syndrome; P_{IA}: Intra-abdominal pressure; P_{IT}: Intrathoracic pressure; P_{TP}: Transpulmonary pressure; LV: Left ventricular; PEEP: Positive end-expiratory pressure; PPV: Arterial pulse pressure variation; RV: Right ventricular; SV: Stroke volume; V_T: Tidal volume; WIPO: Weaning-induced pulmonary edema.

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